Amendments to the Specification:

Please replace the paragraph beginning at page 2 line 4, with the following rewritten paragraph:

C. pneumoniae is a common cause of community acquired pneumonia, only less frequent than *Streptococcus pneumoniae* and *Mycoplasma pneumoniae* (Grayston *et al.* (1995) Journal of Infectious Diseases 168:1231; Campos *et al.* (1995) Investigation of Ophthalmology and Visual Science 36:1477). It can also cause upper respiratory tract symptoms and disease, including bronchitis and sinusitis (Grayston *et al.* (1995) Journal of Infectious Diseases 168:1231; Grayston *et al* (1990) Journal of Infectious Diseases 161:618; Marrie (1993) Clinical Infectious Diseases. 18:501; Wang *et al* (1986) Chlamydial infections[[)]]. Cambridge University Press, Cambridge. p. 329). The great majority of the adult population (over 60%) has antibodies to *C. pneumoniae* (Wang *et al* (1986) Chlamydial infections. Cambridge University Press, Cambridge. p. 329), indicating past infection which was unrecognized or asymptomatic.

Please replace the paragraph beginning at page 4 line 25, with the following rewritten paragraph:

Of considerable importance is the association of atherosclerosis and *C. pneumoniae* infection. There are several epidemiological studies showing a correlation of previous infections with *C. pneumoniae* and heart attacks, coronary artery and carotid artery disease (Saikku *et al.*(1988) Lancet; ii:983; Thom *et al.* (1992) JAMA 268:68; Linnanmaki *et al.* (1993), Circulation 87:1030; Saikku *et al.* (1992) Annals Internal Medicine 116:273; Melnick *et al* (1993) American Journal of Medicine 95:499). Moreover, the organisms ~~has~~ have been detected in atheromas and fatty streaks of the coronary, carotid, peripheral arteries and aorta (Shor *et al.* (1992) South African. Medical Journal 82:158; Kuo *et al.* (1993) Journal of Infectious Diseases 167:841; Kuo *et al.* (1993) Arteriosclerosis and Thrombosis 13:1500; Campbell *et al* (1995) Journal of Infectious Diseases 172:585; Chiu *et al.* Circulation, 1997 (In Press)). Viable *C. pneumoniae* has been recovered from the coronary and carotid artery (Ramirez *et al* (1996) Annals of Internal Medicine 125:979; Jackson *et al.* Abst. K121, p272, 36th ICAAC, 15-18 Sept. 1996, New Orleans). Furthermore, it

has been shown that *C. pneumoniae* can induce changes of atherosclerosis in a rabbit model (Fong *et al* (1997) Journal of Clinical Microbiolology 35:48). Taken together, these results indicate that it is highly probable that *C. pneumoniae* can cause atherosclerosis in humans, though the epidemiological importance of chlamydial atherosclerosis remains to be demonstrated.

Please replace the paragraph beginning at page 6 line 15, with the following rewritten paragraph:

Studies with *C. trachomatis* and *C. psittaci* indicate that safe and effective vaccine against Chlamydia is an attainable goal. For example, mice which have recovered from a lung infection with *C. trachomatis* are protected from infertility induced by a subsequent vaginal challenge (Pal *et al.*(1996) Infection and Immunity.64:5341). Similarly, sheep immunized with inactivated *C. psittaci* were protected from subsequent chlamydial-induced abortions and stillbirths (Jones *et al.* (1995) Vaccine 13:715). Protection from chlamydial infections has been associated with Th1 immune responses, particularly the induction of ~~INFg~~ IFNgamma - producing CD4+T-cells (Igietsemes *et al.* (1993) Immunology 5:317). The adoptive transfer of CD4+ cell lines or clones to nude or SCID mice conferred protection from challenge or cleared chronic disease (Igietseme *et al* (1993) Regional Immunology 5:317; Magee *et al* (1993) Regional Immunology 5: 305), and *in vivo* depletion of CD4+ T cells exacerbated disease post-challenge (Landers *et al* (1991) Infection & Immunity 59:3774; Magee *et al* (1995) Infection & Immunity 63:516). However, the presence of sufficiently high titres of neutralising antibody at mucosal surfaces can also exert a protective effect (Cotter *et al.* (1995) Infection and Immunity 63:4704).

Please replace the paragraph beginning at page 14 line 29, with the following rewritten paragraph:

Polynucleotides encoding homologous polypeptides or allelic variants are retrieved by polymerase chain reaction (PCR) amplification of genomic bacterial DNA extracted by conventional methods. This involves the use of synthetic oligonucleotide primers matching upstream and downstream of the 5' and 3' ends of the encoding domain. Suitable primers are designed according to the nucleotide sequence information provided in SEQ ID Nos: 1 to 10. The procedure is as follows:

a primer is selected which consists of 10 to 40, preferably 15 to 25 nucleotides. It is advantageous to select primers containing C and G nucleotides in a proportion sufficient to ensure efficient hybridization; *i.e.*, an amount of C and G nucleotides of at least 40%, preferably 50% of the total nucleotide content.A standard PCR reaction contains typically 0.5 to 5 Units of ~~Taq~~ Taq™ DNA polymerase per 100 μL, 20 to 200 μM deoxynucleotide each, preferably at equivalent concentrations, 0.5 to 2.5 ~~MM~~ mM magnesium over the total deoxynucleotide concentration, 10^5 to 10^6 target molecules, and about 20 pmol of each primer. About 25 to 50 PCR cycles are performed, with an annealing temperature 15°C to 5°C below the true Tm of the primers. A more stringent annealing temperature improves discrimination against incorrectly annealed primers and reduces incorportion of incorrect nucleotides at the 3' end of primers. A denaturation temperature of 95°C to 97°C is typical, although higher temperatures may be appropriate for dematuration of G+C-rich targets. The number of cycles performed depends on the starting concentration of target molecules, though typically more than 40 cycles is not recommended as non-specific background products tend to accumulate.

Please replace the paragraph beginning at page 16 line 20, with the following rewritten paragraph:

Useful homologs and fragments thereof that do not occur naturally are designed using known methods for identifying regions of an antigen that are likely to tolerate amino acid sequence changes and/or deletions. As an example, homologous polypeptides from different species are compared; conserved sequences are identified. The more divergent sequences are the most likely to tolerate sequence changes. Homology among sequences may be analyzed using the ~~BLAST~~ BLAST™ homology searching algorithm of Altschul et al., Nucleic Acids Res.25:3389-3402 (1997). Alternatively, sequences are modified such that they become more reactive to T- and/or B-cells. (See Figures 11 to 15 below for identification of T- and B- epitopes). Yet another alternative is to mutate a particular amino acid residue or sequence within the polypeptide *in vitro*, then screen the mutant polypeptides for their ability to prevent or treat Chlamydia infection according to the method outlined below.

Please replace the paragraph beginning at page 17 line 15, with the following rewritten paragraph:

By "conferring protection" is meant that there is a reduction ~~is~~ in severity of any of the effects of Chlamydia infection, in comparison with a control animal which was not immunized with the test homolog or fragment.

Please replace the paragraphs beginning at page 17 line 19, with the following rewritten paragraphs:

It has been previously demonstrated (Yang, Z. P., Chi, E. Y., Kuo, C. C. and Grayston, J. T. 1993. A mouse model of *C. pneumoniae* strain TWAR pneumonitis. Infect. Immun. 61(5):2037-2040) that mice are susceptible to intranasal infection with different isolates of *C. pneumoniae*. Strain AR-39 (Chi, E. Y., Kuo, C. C. and Grayston, J. T. , 1987. Unique ultrastructure in the elementary body of Chlamydia sp. strain TWAR. J. Bacteriol. 169(8):3757-63) ~~was~~ is used in Balb/c mice as a challenge infection model to examine the capacity of chlamydia gene products delivered as naked DNA to elicit a protective response against a sublethal *C. pneumoniae* lung infection. Protective immunity is defined as an accelerated clearance of pulmonary infection.

Groups of 7 to 9 week old male Balb/c mice (6 to 10 per group) ~~were~~ are immunized intramuscularly (i.m.) plus intranasally (i.n.) with plasmid DNA containing the coding sequence of a *C. pneumoniae* polypeptide such as CPN100634 (SEQ ID Nos: 1 and 2 encoding SEQ ID No: 11), CPN100635 (SEQ ID Nos: 3 and 4 encoding SEQ ID No: 12 and 13), CPN100638 (SEQ ID Nos: 5 and 6 encoding SEQ ID No: 14), CPN100639 (SEQ ID Nos: 7 and 8 encoding SEQ ID No: 15), and CPN100708 (SEQ ID Nos: 9 and 10 encoding SEQ ID No: 16). Saline or the plasmid vector lacking an inserted chlamydial gene ~~was~~ is given to groups of control animals.

For i.m. immunization alternate left and right quadriceps ~~were~~ are injected with 100μg of DNA in 50μl of PBS on three occasions at 0, 3 and 6 weeks. For i.n. immunization, anaesthetized mice ~~aspirated~~ aspirates 50μl of PBS containing 50 μg DNA on three occasions at 0, 3 and 6 weeks. At week 8, immunized mice ~~were~~ are inoculated i.n. with 5 x 10^5 IFU of *C. pneumoniae,* strain AR39 in 100μl of SPG buffer to test their ability to limit the growth of a sublethal *C. pneumoniae* challenge.

Lungs ~~were~~ are taken from mice at day 9 post-challenge and immediately homogenised in SPG buffer (7.5% sucrose, 5mM glutamate, 12.5mM phosphate pH7.5). The homogenate ~~was~~ is stored frozen at –70°C until assay. Dilutions of the homogenate ~~were~~ are assayed for the presence of infectious chlamydia by inoculation onto monolayers of susceptible cells. The inoculum ~~was~~ is centrifuged onto the cells at 3000rpm for 1 hour, then the cells ~~were~~ are incubated for three days at 35°C in the presence of 1μg/ml cycloheximide. After incubation the monolayers ~~were~~ are fixed with formalin and methanol then immunoperoxidase stained for the presence of chlamydial inclusions using convalescent sera from rabbits infected with *C. pneumoniae* and metal-enhanced DAB as a peroxidase substrate.

Please replace the paragraph beginning at page 21 line 16, with the following rewritten paragraph:

As used herein, a fusion polypeptide is one that contains a polypeptide or a polypeptide derivative of the invention fused at the N- or C-terminal end to any other polypeptide (hereinafter referred to as a peptide tail). A simple way to obtain such a fusion polypeptide is by translation of an in-frame fusion of the polynucleotide sequences, *i.e.*, a hybrid gene. The hybrid gene encoding the fusion polypeptide is inserted into an expression vector which is used to transform or transfect a host cell. Alternatively, the polynucleotide sequence encoding the polypeptide or polypeptide derivative is inserted into an expression vector in which the polynucleotide encoding the peptide tail is already present. Such vectors and instructions for their use are commercially available, *e.g.* the ~~pMal-c2 or pMal-p2~~ pMal-c2™ or pMal-p2™ system from New England Biolabs, in which the peptide tail is a maltose binding protein, the glutathione-S-transferase system of Pharmacia, or the ~~His-Tag~~ His-Tag™ system available from Novagen. These and other expression systems provide convenient means for further purification of polypeptides and derivatives of the invention.

Please replace the paragraph beginning at page 23 line 30, with the following rewritten paragraph:

A recombinant expression system is selected from procaryotic and eucaryotic hosts. Eucaryotic hosts include yeast cells (*e.g.*, *Saccharomyces cerevisiae* or *Pichia pastoris*), mammalian cells (*e.g.*, COS1, NIH3T3, or JEG3 cells), arthropods cells

(*e.g.*, *Spodoptera frugiperda* (SF9) cells), and plant cells. A preferred expression system is a procaryotic host such as *E. coli*. Bacterial and eucaryotic cells are available from a number of different sources including commercial sources to those skilled in the art, *e.g.*, the American Type Culture Collection (ATCC; ~~Rockville, Maryland~~ 10801 University Boulevard, Manassas, VA 20110-2209). Commercial sources of cells used for recombinant protein expression also provide instructions for usage of the cells.

Please replace the paragraph beginning at page 38 line 18, with the following rewritten paragraph:

In an ELISA assay, the product to be screened is preferably used as the coating antigen. A purified preparation is preferred, although a whole cell extract can also be used. Briefly, about 100 µl of a preparation at about 10 µg protein/ml are distributed into wells of a 96-well polycarbonate ELISA plate. The plate is incubated for 2 hours at 37°C then overnight at 4°C. The plate is washed with phosphate buffer saline (PBS) containing 0.05% ~~Tween 20~~ Tween 20™ (PBS/Tween buffer). The wells are saturated with 250 µl PBS containing 1% bovine serum albumin (BSA) to prevent non-specific antibody binding. After 1 hour incubation at 37°C, the plate is washed with PBS/Tween buffer. The antiserum is serially diluted in PBS/Tween buffer containing 0.5% BSA. 100 µl of dilutions are added per well. The plate is incubated for 90 minutes at 37°C, washed and evaluated according to standard procedures. For example, a goat anti-rabbit peroxidase conjugate is added to the wells when specific antibodies were raised in rabbits. Incubation is carried out for 90 minutes at 37°C and the plate is washed. The reaction is developed with the appropriate substrate and the reaction is measured by colorimetry (absorbance measured spectrophotometrically). Under the above experimental conditions, a positive reaction is shown by O.D. values greater than a non immune control serum.